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Pneumatosis intestinalis in a cohort of children with neurological impairment: A patients group with a management dilemma

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Abstract

Introduction: Pneumatosis intestinalis (PI) is uncommon in school-age children. We studied a cohort of neurologically impaired school-age children with PI to formulate an optimum management plan.

Method: We retrospectively studied all school-age children who were referred to paediatric surgeons with radiological evidence of PI identified between 2015 and November 2016. We analysed data on patient demographics, medications, feeding, associated comorbidities, presentation, investigations and treatments.

Main results: neurologically impaired Five patients with a median age of 7 years (range 5-9) were referred for surgical opinion with evidence of PI on their abdominal xrays. Four of the patients had an associated pneumoperitoneum. Interestingly, all patients had cerebral palsy, such that they were significantly neurologically impaired and unable to communicate clearly. Four patients had a laparoscopy/laparotomy at first presentation, with no findings of ischaemic bowel, peritoneal soiling or perforation despite the prescence of pneumoperitoneum on xrays, however, obvious colonic pneumatosis was seen. Four patients were gastrostomy fed, one was jejunally fed. Three patients were medically treated for constipation and two for chronic lung disease (CLD). Four patients had subsequent presentations, which were successfully managed without surgical intervention, despite the prescence of pneumoperitoneum.

Conclusion: In neurologically impaired school-age children, the presence of pneumatosis and pneumoperitoneum does not mandate bowel ischaemia or perforation and therefore could be successfully managed conservatively without the need for surgery.

Keywords: pneumatosis, intestinalis, cohort, children neurological impairment; pneumatosis intestinalis; pneumoperitoneum

Introduction

Pneumatosis intestinalis (PI) refers to the presence of gas within the wall of the small or large intestine. Intramural gas can also affect the stomach, but this condition is referred to as gastric pneumatosis. Since its first description, PI has appeared in the literature under many names, including pneumatosis cystoides intestinalis, intramural gas, pneumatosis coli, pseudolipomatosis, intestinal emphysema, bullous emphysema of the intestine, and lymphopneumatosis.

The pathogenesis of PI is poorly understood, and is probably multifactorial. In some cases, PI is an incidental finding associated with a benign etiology, whereas in others, it portends a life-threatening intra-abdominal condition. As a result of the wide array of clinical settings in which PI is encountered, its implications are often misinterpreted. This topic will review the epidemiology, pathogenesis, clinical features, evaluation, and management of PI. The clinical features of necrotizing enterocolitis, an important cause of PI in newborns, is discussed in detail separately.

Review of Related Literature

This review illustrates the changing paradigms in the understanding of the pathogenesis of pneumatosis intestinalis. Although many theories have been evoked, pragmatically there appear to be four major clinical and diagnostic imaging considerations. The most common and most emergent life-threatening cause of intramural bowel gas is the result of bowel necrosis due to bowel ischemia, infarction, necrotizing enterocolitis, neutropenic colitis, volvulus, and sepsis. In the stomach, intramural gas can be caused by emphysematous gastritis or ingestion of caustic agents. These situations represent surgical emergencies. Pneumatosis is found secondary to mucosal disruption presumably due to over-distention from peptic ulcer, pyloric stenosis, annular pancreas, and even to more distal obstruction. Disruption can also be caused by ulceration, erosions, or trauma, including the trauma of child abuse. Disruption can also be iatrogenic from intracatheter jejunal feeding tubes, stent perforation, sclerotherapy, or surgical or endoscopic trauma. In these cases, the gas may be focal or linear. Treatment depends on the extent of the disruption and the underlying cause. A more subtle form of mucosal disruption may occur due to mucosal erosions and also to defects in intestinal crypts secondary to acute and subclinical enteritides that allow intraluminal bacterial gas under pressure to percolate into the bowel wall layers, particularly the submucosa, Pneumatosis, often linear or cystic in appearance, is seen with increased frequency in patients who are immunocompromised because of steroids, chemotherapy, radiation therapy, or AIDS. In these cases, the pneumatosis may result from intraluminal bacterial gas entering the bowel wall due to increased mucosal permeability caused by defects in bowel wall lymphoid tissue. Clinical and imaging findings are important in the differentiation of this transient pneumatosis from fulminant life-threatening causes in this subset of patients. A pulmonary cause must still be considered in cases of chronic obstructive pulmonary disease, asthma, and cystic fibrosis. It can occur with barotrauma and after chest tube placement. It may relate to increased intrathoracic

pressure associated with retching and vomiting. The possibility remains that occasionally the origin of pneumatosis intestinalis will remain cryptogenic--caused but unexplained.

Research Study

Five children, ages 5 to 9 years, developed pneumatosis intestinalis (PI) after allogeneic bone marrow transplantation (BMT) for acute leukemia or severe aplastic anemia. PI was detected at a median of 48 days (range, 10–63 days) after BMT and was associated with abdominal symptoms and clinical signs. All patients had severe systemic and/or high-grade cutaneous acute graft-versus-host disease (AGVHD) at some time after BMT and were receiving corticosteroids at the time of development of PI; however, PI was associated with concomitant severe AGVHD in only one patient. One patient with PI had*Hafnia alvei* bacteremia and another patient had gastroenteritis due to rotavirus and adenovirus. All patients were treated with supportive care and systemic broad-spectrum antibiotics, and PI resolved 2–16 days after onset. Two patients died with BMT-associated complications unrelated to PI. Multiple factors contribute to the development of PI after BMT, and the prognosis for recovery from PI is good with medical management alone. Overall survival in these patients is dependent on the frequency and severity of other conditions, such as AGVHD and opportunistic infections, after BMT.

Discussion

In our observation more and more benign etiologies for pneumatosis intestinalis are described in the literature, it is very important for the radiologist to understand the pathophysiology of pneumatosis and recognize the findings on different imaging modalities. With appropriate clinical history, the radiologist plays a crucial role in differentiating between medical and surgical causes of pneumatosis intestinalis and suggesting appropriate imaging studies or follow up. the presence of pneumatosis and pneumoperitoneum does not mandate bowel ischaemia or perforation and therefore could be successfully managed conservatively without the need for surgery.

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